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To: The Treasury
Consultation: Ban on the use of adverse genetic tests in life insurance — draft regulations
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Submission on the draft *Insurance Contracts Amendment (Genetic Testing Protections in Life Insurance) Regulations 2026*

Science for All welcomes the opportunity to make this submission. We are an Australian registered charity (ABN 37 636 063 351) that works to involve people and communities in shaping science and research, with a focus on public and community involvement in health and genomic research and on the governance of research data.

We commend the Government on this landmark reform: a complete ban on the use of genetic test results in life-insurance underwriting, without caps or exceptions, will protect Australians from genetic discrimination and remove a documented barrier to participation in health and medical research.[1] The explanatory statement rightly notes that a central purpose is to provide certainty that genetic testing — including *"through participation in health or medical research"* — will not affect a person's life insurance,[2] which is the standpoint from which we make this submission.

We make this submission in our own right, and support the broad thrust of recommendations being made by expert groups associated with the A-GLIMMER project.[3] It draws on peer-reviewed research led by our Director, Dr Jack Nunn, including a scoping review of public involvement in global genomics research[4] and co-design studies with people affected by a rare condition[5] and donor-conceived

[1]Tiller J, Gleeson P, McInerney-Leo AM, et al. Final Stakeholder Report of the Australian Genetics and Life Insurance Moratorium: Monitoring the Effectiveness and Response (A-GLIMMER) Project. Monash University; 2023. doi:10.26180/23564538.
[2]Exposure Draft Explanatory Statement, Insurance Contracts Amendment (Genetic Testing Protections in Life Insurance) Regulations 2026 (Cth), Attachment A (regulation 13A).
[3]A-GLIMMER Final Stakeholder Report, above n 1.
[4]Nunn JS, Tiller J, Fransquet P, Lacaze P. Public involvement in global genomics research: a scoping review. Front Public Health. 2019;7:79. doi:10.3389/fpubh.2019.00079.
[5]Nunn JS, Gwynne K, Gray S, Lacaze P. Involving people affected by a rare condition in shaping future genomic research. Res Involv Engagem. 2021;7:14. doi:10.1186/s40900-021-00256-3.

people,[6] as well as work with older adults and, with collaborators, Aboriginal communities co-designing precision medicine.

Each recommendation is framed as a clarification of concepts the Regulations are empowered to prescribe — the meaning of a genetic test result, a genetic predisposition, and protected genetic information, and the conduct the prohibition covers — and is consistent with the Act's purpose, rather than a free-standing obligation outside the regulation-making power. Suggested drafting is at Appendix A; suggested additions to the predisposition list are at Appendix B.

Summary of recommendations

1. Codify the principle of manifestation in the operative text of the Regulations.

2. Broaden the illustrative list of named predispositions — in particular, add inherited cardiac conditions, which are currently absent (Appendix B).

3. Clarify that polygenic risk scores, pharmacogenomic results, susceptibility (risk) results, and secondary or incidental findings are protected where no related disease has been diagnosed.

4. Clarify that carrier status, including results from reproductive, newborn and population carrier screening, is protected.

5. Confirm that linking protected genetic information to an identifiable individual is itself a prohibited use, while permitting limited compliance records and de-identified, aggregate reporting.

6. Prevent circumvention through family history, or inferred or derived genetic information (including algorithmic risk models), used as a proxy.

Fear of genetic discrimination is a documented barrier to research participation and trust

Concern about insurance discrimination recurs, unprompted, as a barrier to participation in genomic research across diverse Australian communities. In a co-designed study with people affected by a rare condition, participants described it as a *"valid community fear"* that data could be used by insurers, and some parents feared a child would be *"refused insurance because of a decision I have made"* — the parent's own decision to take part in research.[7]

These concerns are consistent with the broader Australian evidence base,[8] and are not limited to single-gene conditions: there is emerging evidence that fear of discrimination may also reduce willingness to undergo polygenic testing and to take part in the research that generates it.[9]

The significance is twofold. The harms the Act seeks to prevent are real and felt across the community, not hypothetical; and the public will judge the ban not only by what insurers do at underwriting, but by whether genetic information is collected, retained, linked, flagged or used indirectly at all. Clarity and comprehensiveness in the Regulations are therefore essential to the trust on which the reform's benefits depend.

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[6]Nunn JS, Crawshaw M, Lacaze P. Co-designing genomics research with a large group of donor-conceived siblings. Res Involv Engagem. 2021;7:89. doi:10.1186/s40900-021-00325-7.
[7]Nunn et al. (rare condition), above n 5.
[8]See, e.g., Tiller J, Winship I, Otlowski MF, Lacaze PA. Community concerns about genetic discrimination in life insurance persist in Australia: a survey of consumers offered genetic testing. Eur J Hum Genet. 2023.
[9]Yanes T, Tiller J, Haining CM, Wallingford C, Otlowski M, Keogh L, McInerney-Leo A, Lacaze P. Future implications of polygenic risk scores for life insurance underwriting. npj Genom Med. 2024;9:25. doi:10.1038/s41525-024-00407-x.

Recommendation 1 — Codify the principle of manifestation

The Act removes from protection information about a disease for which a person *"has been clinically diagnosed"*, so the ban turns on the boundary between a genetic predisposition and a clinically diagnosed disease. Because the statutory test relies heavily on the concept of diagnosis itself, a predisposition described using disease terminology could be mischaracterised as a diagnosis. The explanatory statement already supplies the right principle — it treats a person clinically diagnosed with Huntington disease *"based on medical assessment and the manifestation of symptoms"* as outside protection, while the predisposition remains protected[10] — but states it only in the explanatory material, not the operative text.

"Manifestation" is already used in cognate areas of Australian law: the New South Wales Court of Appeal has distinguished a genetic predisposition from the condition itself, noting it *"might never manifest itself"*,[11] and the same distinction underlies the United States Genetic Information Nondiscrimination Act ("manifested disease"). The principle should be placed in the Regulations so it governs interpretation directly (Appendix A). If a directly analogous Australian life-insurance authority is available, it should be substituted, as Treasury may find an insurance precedent more persuasive.

Recommendation 2 — Broaden the illustrative list, and add inherited cardiac conditions

Of the fourteen entries at regulation 13A(1), eleven are hereditary cancer syndromes; the rest are one lipid disorder, one neurodegenerative condition and one haematological condition. Although regulation 13A(2) makes the list non-exhaustive, its composition invites the inference that conditions from absent categories are treated differently — the same risk as Recommendation 1, sharper for categories the list omits entirely. Inherited cardiac conditions are the clearest example: hypertrophic, dilated and arrhythmogenic cardiomyopathies, long QT syndrome, Brugada syndrome and catecholaminergic polymorphic ventricular tachycardia are typically autosomal-dominant with incomplete penetrance and frequently produce *"genotype-positive, phenotype-negative"* individuals who carry a variant but have no clinical disease.[12] They are a major indication for pre-symptomatic testing, and such individuals perceive their risk as low pre-test in the absence of symptoms.[13]

We recommend adding representative examples from the absent categories (Appendix B). Haemochromatosis is a clear Australian example: the C282Y genotype is common (around 1 in 200 people of Anglo-Celtic descent) yet clinical penetrance is low and contested, so it is routinely conflated with the disease even in asymptomatic people.[14] No list can be exhaustive; the durable safeguard remains Recommendation 1.

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[10]Explanatory statement, above n 2.

[11]*Matthew Hall Pty Ltd v Smart* [2000] NSWCA 284.

[12]Musunuru K, et al. (American Heart Association). Genetic Testing for Inherited Cardiovascular Diseases: A Scientific Statement. Circ Genom Precis Med. 2020. doi:10.1161/HCG.0000000000000067.

[13]Ormondroyd E, Oates S, Parker M, Blair E, Watkins H. Pre-symptomatic genetic testing for inherited cardiac conditions: a qualitative exploration of psychosocial and ethical implications. Eur J Hum Genet. 2014;22(1):88–93. doi:10.1038/ejhg.2013.81.

[14]Allen KJ, Gurrin LC, Constantine CC, et al. Iron-overload-related disease in HFE hereditary haemochromatosis. N Engl J Med. 2008;358:221–230.

Recommendation 3 — Clarify that further categories of genetic information are protected

The draft focuses on single-gene predispositions. Other forms of genetic test information are now in wide use and indicate risk rather than diagnosis, and future underwriting may attempt to use them. The Regulations should clarify that the following are protected where no related disease has been clinically diagnosed: (a) polygenic (genomic) risk scores, which estimate susceptibility to common diseases and are expected to become significant in underwriting;[15] (b) pharmacogenomic results (e.g. CYP2D6, CYP2C19, TPMT, DPYD), which are not diagnoses but may be used to infer health status; (c) single-gene results indicating susceptibility or increased risk rather than diagnosis (e.g. HLA-B27, HLA-DQ2/DQ8, the APOE ε4 allele); and (d) secondary or incidental findings identified during testing for another purpose (Appendix A).

Recommendation 4 — Clarify the protected status of carrier status, including screening

A person may carry a single variant for a recessively or X-linked condition (such as cystic fibrosis or spinal muscular atrophy) without being at risk of developing it. There is limited evidence that such carriers have faced insurance discrimination to date, and a reasonable community expectation that they will be protected — reinforced by Australia's investment in reproductive carrier screening (Mackenzie's Mission) and the November 2023 Medicare listing of carrier screening for cystic fibrosis, spinal muscular atrophy and fragile X syndrome.[16] A carrier has nothing to "manifest", so this is best stated directly; and because some carrier states are not asymptomatic (e.g. Fragile X premutation carriers, some female X-linked carriers), the matter is better addressed by clear principle. Carrier status — including results from reproductive, newborn or population screening — should be protected and not usable as a proxy (Appendix A).

Recommendation 5 — Confirm that linking protected information to an individual is a prohibited use

Public confidence depends not only on underwriting use but on whether genetic information is collected, retained, linked or flagged at all; research shows the prospect of such data being held and potentially used to a person's or group's detriment is itself a barrier to participation and trust.[17] There are indications insurers are considering mechanisms to identify applicants known to have had genetic testing.[18] To remain within the regulation-making power, we frame this as preventing indirect use rather than a new information-management regime: creating a flag or record that links protected information to an identifiable person, and acting on it, is a form of "use" and should be confirmed to fall within the prohibition (Appendix A).

A narrow exception is needed so insurers can still identify and remove inadvertently disclosed information and demonstrate compliance. So the prohibition is enforceable without itself creating a tracking risk, insurers could be required to keep auditable evidence of compliance and provide the

[15]Yanes et al., above n 9.

[16]Mackenzie's Mission (the Australian Reproductive Genetic Carrier Screening Project), administered by Australian Genomics; reproductive genetic carrier screening for cystic fibrosis, spinal muscular atrophy and fragile X syndrome was added to the Medicare Benefits Schedule in November 2023.

[17]Nunn et al. (rare condition), above n 5.

[18][Citation to be inserted — industry commentary published since the Act's passage (e.g. the Re(in) Asia commentary featuring Jessica Thurtell, Clyde & Co, 2026), as referenced in submissions by the A-GLIMMER group.]

regulator with aggregate, de-identified reporting — through the Regulations or ASIC guidance — using standardised, machine-readable formats. Open standards exist for this kind of reporting; for example, STARDIT is already used to report initiatives by organisations including Cochrane and Australian Genomics.[19] (We disclose that our Director co-developed STARDIT; we note it only as one open standard among several.)

Recommendation 6 — Prevent circumvention through family history and inferred information

Once direct use is prohibited, the obvious avenues for circumvention are indirect. The Act bans use of genetic test results, but insurers may still collect family history and may increasingly infer genetic risk from family history, medical records, ancestry data or algorithmic (AI) models. Because genetic discrimination is differential treatment on the basis of *real or assumed* genetic characteristics,[20] protection should extend to information that functions as a proxy. We recommend the Regulations ensure family-history questions cannot be used as a proxy for prohibited genetic information, and provide that information derived from, inferred from, or predictive of a person's genetic characteristics receives equivalent protection where it substitutes for a protected genetic test result (Appendix A). **This should expressly include clinical or screening findings that, while not themselves a clinical diagnosis, suggest or allow inference of a protected genetic variant**. Inherited cardiac conditions show why this matters: pre-symptomatic cardiac screening — an electrocardiogram or cardiac imaging — can reveal subclinical features in a person with no clinical disease that point to an underlying pathogenic variant. Such screening is potentially life-saving, identifying people at risk of sudden cardiac death in time to intervene[21]; yet if its findings could be used by insurers as a proxy for the protected genetic result, people may avoid screening, at a direct cost to lives.

Conclusion

Science for All strongly supports the draft Regulations. The amendments we propose are clarifications within the regulation-making power, directed at the certainty, comprehensiveness and durability of the protection and the public trust on which its benefits depend. We would welcome the opportunity to provide further evidence and to assist in communicating the protections to the public and the research community.

We consent to this submission being published.

Yours sincerely,

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[19]Nunn JS, Shafee T, Chang S, et al. Standardised data on initiatives—STARDIT: Beta version. Res Involv Engagem. 2022;8(1):31. doi:10.1186/s40900-022-00363-9.

[20]Yanes et al., above n 9.

[21] MacLachlan H, Bhatia R, Raju H, Mellor G, Androulakis E, Basu J, Behr ER, Chandra N, Chatrath N, Dhutia H, D'Silva A, Fyyaz S, Gati S, Malhotra A, Marwaha S, Miles C, Millar L, Prakash K, Kondapally Seshasai SR, Sheikh N, Specterman M, Tome M, Zaidi A, Finocchiaro G, Sharma S, Papadakis M. Cardiac Screening for Conditions Associated With Sudden Cardiac Death: Yield, Interventions, and SCA/SCD Incidence in 104,369 Young Individuals. J Am Coll Cardiol. 2026 Feb 24;87(7):756-768. doi: 10.1016/j.jacc.2025.11.049. PMID: 41738673.

Appendix A — Suggested drafting

The following additions to regulation 13A would give effect to Recommendations 1 and 3–6. (Recommendation 2 is given effect by adding the conditions in Appendix B to regulation 13A(1).)

Recommendation 1. *A genetic predisposition that has not manifested as a disease for which the individual has received a clinical diagnosis (within the meaning of relevant parts of the Act) is not itself a clinical diagnosis, and information about it is protected genetic information. A subclinical sign or screening finding that falls short of a clinical diagnosis does not constitute manifestation*

Recommendation 3. *To avoid doubt, each of the following is protected genetic information where the individual has not received a clinical diagnosis of a related disease: (a) a polygenic or genomic risk score; (b) a pharmacogenomic result indicating medication response, metabolism or risk of adverse drug reaction; (c) a result indicating susceptibility to, or increased risk of, a condition (as distinct from its clinical diagnosis); and (d) a secondary or incidental finding identified in the course of testing conducted for another purpose.*

Recommendation 4. *To avoid doubt, being a carrier of a genetic variant associated with a recessively or X-linked inherited condition (including a result generated through reproductive, newborn or population carrier screening), where the individual does not have a clinical diagnosis of that condition, is protected genetic information.*

Recommendation 5. *A mechanism, notation, flag, marker, record or process that links a genetic test result, or information derived from it, to an identifiable individual constitutes use of that result for the purposes of the prohibition, and is not permitted except where strictly necessary for compliance with the Act, audit, or the correction or deletion of information that has been inadvertently disclosed. Insurers should maintain auditable evidence of compliance and provide the regulator with aggregate, de-identified reporting.*

Recommendation 6. *An insurer must not use family history, or information derived from, inferred from, or predictive of a person's genetic characteristics (including through algorithmic or statistical models), as a proxy for a genetic test result that is protected under the Act; such information receives equivalent protection where it functions as a substitute for protected genetic information.*

Appendix B — Suggested additions to the predisposition list (regulation 13A(1))

Representative examples from categories not currently reflected in the list. The list is illustrative, not exhaustive; the durable safeguard is the manifestation principle in Recommendation 1.

Category	Suggested additions
Inherited cardiac conditions (currently absent)	Hypertrophic cardiomyopathy; dilated cardiomyopathy; arrhythmogenic (right ventricular) cardiomyopathy; long QT syndrome; Brugada syndrome; catecholaminergic polymorphic ventricular tachycardia
Heritable aortopathies and connective-tissue conditions (currently absent)	Marfan syndrome; Loeys–Dietz syndrome; vascular Ehlers–Danlos syndrome; familial thoracic aortic aneurysm and dissection
Further hereditary cancer syndromes	Hereditary diffuse gastric cancer (CDH1); PTEN hamartoma tumour syndrome (Cowden syndrome); Peutz–Jeghers syndrome; multiple endocrine neoplasia types 1 and 2; hereditary paraganglioma–phaeochromocytoma syndrome; familial melanoma (CDKN2A); hereditary retinoblastoma
Other predispositions with disease-like names (currently absent)	Hereditary haemochromatosis (HFE); autosomal dominant polycystic kidney disease; hereditary transthyretin amyloidosis; alpha-1 antitrypsin deficiency; beta thalassaemia
Neurogenetic conditions beyond Huntington disease	Spinocerebellar ataxias; Friedreich ataxia; myotonic dystrophy; CADASIL; familial / early-onset Alzheimer disease; familial frontotemporal dementia and motor neurone disease